Correspondence

The Editorial Board will be pleased to receive and consider for publication correspondence containing information of interest to physicians or commenting on issues of the day. Letters ordinarily should not exceed 600 words, and must be typewritten, double-spaced and submitted in duplicate (the original typescript and one copy). Authors will be given an opportunity to review any substantial editing or abridgement before publication.

Shoulder Pain: Differential Diagnosis

TO THE EDITOR: The review by White in the October issue[1] outlines the general approach to the differential diagnosis of shoulder pain. Emphasis is placed on the description and treatment of local musculoskeletal conditions affecting the rotator cuff. Much less attention is given to a "variety of disorders" producing pain in the vicinity of the shoulder (cervical spine, neurologic dysfunction, neoplasia and others). Yet, a significant proportion of patients have neck-related pain as disclosed in a recent retrospective study of "shoulder pain" at our institution. Therefore, close attention to the evaluation of the cervical spine and the trapezial muscles, followed by a thorough examination of the rotator cuff, should yield diagnoses in 80 percent of cases. Having excluded these common conditions, one can rationally proceed in the evaluation of the intrinsic bone, joint or nerve processes (see Table 1 below).

TABLE 1.—*Differential Diagnosis of Shoulder Pain in 101 Patients Seen During 18 Months (Screened to Exclude Trauma Cases)*

Diagnoses	*Incidence (percent)*
Rotator cuff lesions	
Supraspinatus tendinitis/bursitis	51
Adhesive capsulitis	10
Reflex sympathetic dystrophy	1
Rotator cuff tears	1
Neck-related disease	
Trapezial muscle origin	17
Cervical radiculopathy	4
Biceps tendon lesions	
Tendinitis	2
Rupture	1
Acromioclavicular disease	
Osteoarthritis	1
Ligamentous strain	1
Partial separation	2
Scapulocostal syndrome	4
Glenohumeral disease	
Osteoarthritis	..
Rheumatoid arthritis	1
Arthralgia	1
Septic arthritis	..
Primary bone lesions	..
Miscellaneous	
Pectoralis strain	2
Polymyalgia rheumatica	1
Unknown or unclassifiable	2
TOTAL	102

Therapeutically, we disagree with the author's recommendation of immobilization of a painful shoulder for several days before initiating physical therapy (pendulum exercises). This places a subset of patients (perhaps as many as 25 percent) with acute supraspinatus tendinitis/bursitis at risk for adhesive capsulitis developing. This is especially true of a patient with a low pain threshold. We would strongly urge early corticosteroid injection and subsequent daily pendulum exercises for moderately severe to severe rotator cuff tendinitis/bursitis.

A prospective study of the efficacy of physical therapy, oral administration of phenylbutazone and local injection of corticosteroid in the treatment of supraspinatus tendinitis/bursitis is currently underway at The Oregon Health Sciences University.

BRUCE C. ANDERSON, MD
Northwest Permanente, PC
Portland

SAM KAYE, MD
Third-Year Resident
Primary Care Medicine
The Oregon Health Sciences University
Portland

REFERENCE

1. White RH: Shoulder pain (Topics in Primary Care Medicine). West J Med 1982 Oct; 137:340-345

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TO THE EDITOR: I read Dr Richard White's October article on shoulder pain[1] with interest, and agree that in most patients "neurological" causes of pain in or about the shoulder can be suspected on the basis of symptoms and physical findings. I would like to remind the readers of *WJM* of a neurological syndrome that is often confused with musculoskeletal causes of shoulder pain by primary care physicians, especially early in its course.

Acute brachial neuritis, also known as brachial plexus neuropathy or neuralgic amyotrophy,[2] usually affects healthy young or middle-aged men. The typical patient first notes sudden, intense, unilateral shoulder

pain, most often described as "sharp," "stabbing," "aching" or "gnawing." This pain is not increased by Valsalva maneuver but is often exacerbated by motion of the shoulder. About a third of patients complain of paresthesias in the affected extremity. The pain lasts two or three weeks, during which the patient invariably seeks medical attention; as the pain improves, it is replaced by patchy weakness and atrophy of muscles innervated by the brachial plexus (most often the upper roots). The degree of atrophy and weakness is often profound and exceeds that seen in cervical radiculopathies.

Recovery of motor function is generally satisfactory but may take many months or years. The cause of brachial plexus neuropathy is not known; its occurrence following the administration of foreign sera or vaccines has led to speculation about an immune-mediated pathogenesis. There is no known effective treatment except physical therapy where indicated to prevent the consequences of prolonged joint immobility.

Failure to recognize the syndrome can lead to unnecessary cervical myelography or other invasive procedures, and incorrect diagnoses ranging from musculoskeletal conditions to motor neuron disease.

JAMES W. SCHMIDLEY, MD
Department of Neurology
University of California, San Francisco, School of Medicine
San Francisco

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2. Tsairis P, Dyck PJ, Mulder DW: Natural history of brachial plexus neuropathy. Arch Neurol (Chicago) 1972 Aug; 27:109-117

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TO THE EDITOR: White's nice review of common shoulder pain syndromes appropriately stresses the basic principles of accurate diagnosis and conservative treatment directed at early mobilization of the joint.[1] I believe a word of caution is warranted concerning his advice to treat "acutely inflamed shoulders" with injections of steroids. The author lists septic arthritis in his differential diagnosis of shoulder pain, but does not discuss this relatively uncommon condition. Infection of the shoulder may also involve the subdeltoid bursa. It occurs usually in patients with chronic illness or previous hospital admission but can appear very much like acute bursitis or tendinitis.[2]

I therefore recommend an attempt at aspiration of an inflamed shoulder, including both subdeltoid bursa and shoulder joint. If any fluid is retrieved, Gram stain and culture are advisable, with at least the former preceding any steroid injection that may compromise diagnosis and treatment of occult shoulder infection.

PETER G. TRAFTON, MD
Department of Orthopaedic Surgery
San Francisco General Hospital Medical Center
San Francisco

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2. Gelberman RH, Menon J, Austerlitz MS, et al: Pyogenic arthritis of the shoulder in adults. J Bone Joint Surg [Am] 1980; 62(4):550-553

Separation of Hypereosinophilic Syndrome From Acute Lymphoblastic Leukemia With Reactive Eosinophilia

TO THE EDITOR: In your September 1982 issue, Bachhuber and co-workers[1] presented a case of acute lymphoblastic leukemia with reactive eosinophilia. Their discussion concerning the diagnosis becomes less clear when subsequent clinicopathologic data are presented in following this patient from his original presentation at The Oregon Health Sciences University in April 1980 to autopsy in November 1980 at Sacred Heart Hospital in Eugene, Oregon.

Before the death of the patient, signs of congestive heart failure developed with gated cardiac output studies showing global hypokinesia. The mechanism of death was a cardiac arrhythmia. Autopsy showed an endocardiomyopathy with pronounced endocardial thickening due to fibrosis with eosinophilic infiltration at the base. This process extended into the mitral and aortic valves. Mural thrombi were present in the right and left ventricles. Eosinophilic infiltration also involved the lungs, liver and spleen. The bone marrow showed an increase in cellularity associated with a moderate increase in mature appearing eosinophils.

The original bone marrow study at The Oregon Health Sciences University in mid-October showed the blasts to have myeloid differentiation in the biopsy, with questionable lymphoid differentiation in the touch preparation. There was no maturation arrest in any cell lines. Chomosome markers showed abnormal cytogenetics.

A diagnosis of acute lymphoblastic leukemia with reactive eosinophilia is inconsistent with the clinicopathological data.[2] The pathologic findings at autopsy were completely in keeping with the hypereosinophilic syndrome.[3] Chromosomal aneuploidy has been reported with the hypereosinophilic syndrome.[4] The patient's diagnosis is a collection of clinical findings and, therefore, should be labeled a syndrome rather than a disease.[5]

H. STUART MARKWELL, MD
ED WILSON, MD
McKenzie-Willamette Hospital
Springfield, Oregon

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2. Catovsky D, Bernasconi C, Verdonck PJ, et al: The association of eosinophilia with lymphoblastic leukaemia or lymphoma: A study of seven patients. Br J Haematol 1980 Aug; 45:523-534
3. Chusid MJ, et al: The hypereosinophilic syndrome: Analysis of fourteen cases with review of literature. Medicine 54:1-27
4. Bitran JD, Rowley JD, Plapp F, et al: Chromosomal aneuploidy in a patient with hypereosinophilic syndrome: Evidence for a malignant disease. Am J Med 1977 Dec; 63:1010-1014
5. Fauci AS, et al: The idiopathic hypereosinophilic syndrome. Ann Intern Med 1982 Jul; 97:78-92

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Dr Fitchen Replies

TO THE EDITOR: Without knowledge of the bone marrow findings, the hypereosinophilic syndrome (HES) was obviously an important consideration in my analysis of this case and a possibility that weighed heavily in my